*Conclusions.*—From what has preceded the following conclusions seem justifiable:—

1. Transitory albuminuria is not uncommon in the course of malarial fevers, and is due to the intense visceral congestions characteristic of these affections. It only may endure throughout the height of the congestion, recurring with each return of this, or it may persist in the intervals, in which event a higher grade of congestion is attained, more nearly approaching a condition of acute inflammation.
2. In a proportion of cases, varying with locality and type of prevailing epidemic, or individual conditions, inflammation of the kidney occurs, accompanied by dropsy and the usual symptoms of nephritis.
3. The usual form of malarial nephritis is the tubal and diffuse variety. In this the inflammation seems to be most intense in the vicinity of the glomeruli.
4. Contracted kidney may occur as an advanced stage of malarial nephritis either from long-continued or frequently repeated attacks of malarial fever, or from fibrotic changes such as may ultimately occur in ordinary tubal or diffuse nephritis. It is altogether improbable that this form of malarial renal disease ever occurs primarily as purely interstitial nephritis.
5. These changes may be induced by any form of malarial fever, though they more commonly follow chronic intermittent fever.
6. The tendency of malarial inflammation of the kidney is toward recovery. But from the persistence of the impaludism or the intensity of the inflammation, structural changes may be produced that are characteristic of chronic Bright's disease, when the gravity of the affection will be as that from chronic Bright's disease from whatever cause.
7. Treatment should be directed primarily against the malarial intoxication, more especially in recent cases. A correction of this will often be followed by a complete, though often gradual, subsidence of the nephritis. Even in more chronic cases, the malarial factor in the process should definitely be destroyed if possible, after which the disease should be treated as ordinary Bright's disease.

## ARTICLE XII.

## ACUTE YELLOW ATROPHY, RED ATROPHY, AND HYPERTROPHIC CIRRHOSIS OF THE LIVER.[1]

By J. H. MUSSER, M.D., Chief of the Medical Dispensary of the Hospital of the University of Pennsylvania, Pathologist to the Presbyterian Hospital

THERE are, perhaps, no diseases of the liver upon which there has been more discussion and speculation, than those named at the head of this paper. Of rare occurrence, obscure in etiology, variable in symptoms, it is not surprising that they have excited so much interest. For these reasons no further apology will be required for presenting these studies to your consideration to-night.

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[1] Read before the Pathological Society of Philadelphia, March 13, 1884.

In connection with acute yellow atrophy the clinical history of a case will be given very briefly, the appearances found after death described, and the histological changes discussed. Your attention will be especially invited to the determination of the question whether the degenerative changes are primary, or are secondary to an inflammatory process. After considering cirrhosis of the liver in general, and recording a case of "hypertrophic" cirrhosis, a description of the microscopical appearances of the liver will be detailed, the proper relation of this disease in nosology discussed, while the precise value of the histological changes, will be determined.

Case I. *Acute Yellow Atrophy of the Liver.*[1]—A married woman, twenty-five years old, in the fifth month of her second pregnancy. In her former labour had puerperal convulsions. The presence of this pregnancy was attended by extreme mental depression on account of fear, to such a degree that she resorted to means to provoke abortion. The decided symptoms of acute yellow atrophy of the liver, which lasted six days, were preceded, by some weeks, by a slight catarrhal jaundice. It is rather irrelevant to detail any more of the clinical history, and therefore at once brief notes of the autopsy will be given. The tissues were bile-stained, ecchymoses under the skin were abundant, and the blood was liquid and of a dark mahogany-red colour. The abdominal organs alone were examined. The spleen was slightly enlarged. The kidneys were enlarged; capsule removed easily; stellate veins and capillary circlets enlarged; tissue bile stained and congested; cortex enlarged. On microscopical examination the organ presented the changes of chronic desquamative nephritis with congestion. The liver was found high up and against the vertebral column, separated from the anterior thoracic wall by intestines. It was about one-half its natural size, very soft and flabby; its capsule was wrinkled. Large areas of the external surface were of a mahogany-red, and larger, a yellow colour. On section, the red portions looked not unlike the blood-red coloured pulp of oranges we often see; the yellow were of the characteristic ochre or rhubarb shade. The red appeared congested, the yellow anæmic. In the latter areas the outlines of the lobules were effaced, although the site of the hepatic veins were shown by dark-red puncta. The gall-bladder was small and contained a teaspoonful of olive-green bile. The ducts were empty and patulous. The report further reads that on microscopical examination, "in some areas, the cells of the lobules, were not much degenerated, but all contained one or more oil-globules. In most lobules, however, the cells of the periphery were markedly changed; globules of oil in abundance, shrivelled cells, free nuclei, and numerous lymphoid cells replaced the normal liver cells. Some of the lobules were entirely destroyed. In some a marked infiltration of lymphoid cells in their periphery was noted. The portal vein appeared normal; the hepatic showed cell infiltration. The walls of the hepatic artery were thickened. The hepatic ducts were filled with epithelial cells and the walls thickened. No crystals of leucin or tyrosin were found, but here and there hæmatoidin.

The above is the description of the microscopical picture as seen when the report of the case was made. Lately the sections have been studied again very carefully, and some fuller statements can be made. Thus in some fields, in fact in the larger number of instances, the cells were less affected in the portal than in the hepatic zone of the lobule, although the infiltration of leucocytes was much greater in the former area. This infiltration was so excessive that it amounted almost to a new formation of young connective tissue. In some of the intra-lobular meshes blood corpuscles as well as leucocytes were seen. In some areas the destruction of liver cells had not begun; in others they showed cloudy swelling. The assertion above, "that the portal veins appeared normal, the hepatic showed cell infiltration," was a misplacement of terms, and should be so changed that hepatic would take the place of portal.

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[1] See Trans. Phila. Co. Med. Soc. 1882–1883.

It may be asked what is the relation of this enormous accumulation of lymphoid elements? Is it the primary, and the degenerative the secondary change? Is, in other words, the disease of the liver a diffused inflammation, or is it a fatty degeneration? This is the ground upon which many battles have been fought; in fact the war is still waging; the question is still unsettled. In the report of the above case it was said that the changes were due to a diffused hepatitis. And so at first blush one would think. That such a view is not tenable, the following proposition would seem to uphold.

It is well known that the liver, of all organs save the spleen, is most distensible without its structure being affected, and the cells can undergo a high degree of pressure without degenerative changes in them. Now the amount of exudation is so slight in acute yellow atrophy that it would not be possible to have deleterious influence on the cells; hence one would infer their primary destruction. The liver is highly anæmic in appearance to the naked eye, and a study of a section, as in the above cases, shows an absence of blood in the vessels—sufficient cause, with the primary blood-poison, to excite the acute necrosis. That the necrosis is the primary change is all the more likely when it is remembered that the only plausible explanation for the occurrence of exudation in a bloodless liver is because of the removal of the support of the vessels, and resistance to the blood-pressure by the destruction of the cells. A further reason for the degenerative process being primary may be found in the fact that a similar process takes place in other organs of the body, notably the heart and kidneys. Finally, the histological appearances of the two distinct portions of the liver seem to indicate their sequence to the destruction of the liver-cells. The yellow-coloured portion has been described; sections of the red show greater destruction of the liver-cells, a greater thickening of stroma, and a preponderance of cell-infiltration. Within the lobules there is a network of connective tissue, the meshes of which are filled with granular *débris*—pigment granules and nuclei. Legg has shown that the amount and degree of the red atrophied portion depend entirely upon the duration of the disease, and all authorities agree that it represents an older disorganization than the yellow portion. For these reasons, therefore, it is no doubt true that the fatty degeneration precedes any cell-proliferation; that whatever degree of cell-proliferation is present, it occurs secondarily, and the amount of it depends upon the extent of the primary necrosis.

Many observers call attention to the presence of new ducts, and some to a "regeneration of the liver-tissue taking place from the bile ducts."[1] These appearances were not noted in the above case. The latter statement is so unphysiological that the condition is not likely to have a pathological existence. Reference will be made to the presence of these ducts when speaking of hypertrophic cirrhosis. Bacteria are said to have been found in this disease, and to one species—the Microsporon septicum—has been attributed all the trouble. Dr. Formad called my attention to the presence of colonies of micrococci in the veins of some of the sections; otherwise they would have been overlooked.

*Red Atrophy of the Liver.*—Some confusion appears to exist at the present time regarding this affection. Formerly it was applied to an atrophic degeneration of the liver, following organic heart disease, an advanced nutmeg-congestion. Now the term is applied to the latter stages of acute yellow atrophy,[2] while brown or chronic atrophy is the name given to the liver of chronic heart disease. The writer presented to the society the only specimen of this disease that has been reported. The rarity of the affection is sufficient apology for recurring to it.

Case II.—The organ was diminished to two-thirds its size, weighing one pound thirteen ounces; the left lobe was somewhat enlarged, however. The tissue was firm on pressure, unevenly yellow-tinged, and on section was of a nutmeg appearance. On microscopical examination, the atrophy was more marked around the hepatic venules, where the liver-cells were replaced by a mass of reddish *débris*. At the periphery of the lobules the cells were much less affected, while the increase of connective tissue was marked, both of old and young.

Save the absence of fat globules in the section, the picture much resembles one of acute yellow atrophy. The clinical history forbids any conclusion of such character[3]

In addition, the hepatic vein is always found dilated, while the intralobular capillaries are much increased in size—conditions not present in yellow atrophy.

*Hypertrophic Cirrhosis.*—Cirrhosis of the liver was formerly limited to that chronic inflammation of the connective tissue which accompanied the portal vessels in their distribution, and which was inter-lobular. In addition to "multi-lobular" or "alcoholic" or common cirrhosis, we now include under the term cirrhosis "biliary" or "mono-lobular" or "hypertrophic" cirrhosis, and syphilitic cirrhosis (congenital). Described first by some German observers,[4] attention was only directed to hypertrophic cirrhosis after the investigations of Charcot and Gombault and of Wickam

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[1] Legg on the Bile, etc., p. 512, Report of Waldeyer's Case.

[2] See Goodhart, Atlas of Pathology, Liver Diseases, Sydenham Society Trans.

[3] See Path. Trans., vol. x. p. 95.

[4] See Ziemssen, vol. ix. p. 211.

Legg. It is called "biliary" cirrhosis because the condition is supposed to follow an obstruction to the bile-ducts higher up in their course or at their terminations. The gentleman just mentioned produced the characteristic changes by ligating the ducts of animals, while they assert that similar changes are found in obstruction of the common duct or chronic catarrh of the finer ramifications of the ducts. It is called "mono-lobular" cirrhosis, in contradistinction to multi-lobular cirrhosis, because, while in the latter islets of lobules are inclosed in the connective tissue, in the former single lobules are thus inclosed, while the lobules themselves are invaded by the new growth (intra-lobular), causing atrophy and often complete destruction of the hepatic cells. It is called hypertrophic cirrhosis because the liver is invariably found enlarged.

The following case, which has never been reported, is worthy of study in this connection. The abstract of the clinical record of Dr. Hughes is first given:—

Case III.—This case was admitted to the University Hospital in October, 1882. A married woman, thirty-nine years old, intemperate (malt liquors), of bad moral character, free from syphilis as far as her testimony determined, without knowledge of her family history, of previous good health, noticed, one year before admission, gradual enlargement of the abdomen. She had previously had for years dyspeptic attacks. She did not know when the enlargement began, and had with it dull hepatic pain. Dyspeptic symptoms increased, and the past eight months she vomited after meals. Appetite poor for many months. Lost flesh and strength; had epistaxis occasionally; abdominal and hepatic pains continued, menstruation ceased six months. During the two months before admission, slight jaundice developed and increased in intensity, and yellow vision was observed. When admitted she was jaundiced, and the arterioles of her cheeks injected. Tongue red on edges, dry and brown on dorsum; sordes; constant thirst; poor appetite; weight, fulness, and pain after eating, with acid bilious vomiting at times. Diarrhœa for two days; formerly constipation. Ascites; enlarged liver (third interspace to two inches below ribs), hard, surface rough, edges rounded. Enlarged superficial veins. Urine dark; sp. gr. 1015; contains albumen and granular casts and bile-pigment. She died the sixth day after admission, having been semi-comatose forty-eight hours before death. The first day of admission, twenty-seven and one-half pints of fluid were removed from the abdomen by paracentesis.

*Hypertrophic Cirrhosis of the Liver.*—This abstract of the notes of the autopsy, three and a half hours after death, is taken from the record in the University Hospital.

*External Examination.*—No rigor mortis; intense jaundice; the whole body of uniform deep orange-yellow tinge; sclerotic coats of eyes also deep yellow; pupils widely dilated; marked discoloration of the whole posterior part of body; no emaciation of body; adipose tissue well preserved, or rather in excess, and of unusual soft and flabby character; abdomen greatly distended with ascitic fluid; no œdema elsewhere.

*Thorax.*—Heart and pericardium normal. Lungs normal. Some right-sided chronic pleurisy, with a few slight adhesions, both diaphragmatic and parietal, and with moderate effusion on corresponding side.

*Abdomen.*—Eight quarts of a deep-yellow, perfectly clear fluid in peritoneal cavity. Omentum normal, but with enormous deposition of fat.

*Liver* protruding five inches below lower border of ribs; very marked perihepatitis, with strong adhesions; the organ is uniformly and much enlarged (about one-third); weight, with pancreas together, 3000 grammes. Surface rough, granular, like in ordinary cirrhosis; colour deep orange-yellow, and uniform on section; consistence firm, but not as hard as usual in cirrhosis; the parenchyma is rather elastic; gall-bladder distended and filled with a thin, mucous, pale-yellow, viscid fluid, substituting the bile; gall-duct nearly obliterated—a probe could be passed through it only with great difficulty; the mucous membrane of gall-bladder and duct is thickened and the seat of catarrhal inflammation. (See Microscopic Report.)

*Spleen* enlarged, congested, tough in consistence; weight, 265 grammes.

*Kidneys* presented the appearance of chronic catarrhal nephritis.

*Microscopic Appearances of the Liver.*—The connective tissue predominates in quantity (bulk) over the liver-cells. This large increase is mainly intra-lobular, or rather inter-*cellular*, separating widely each individual liver-cell from the other in nearly the same degree as the lobules are separated by the likewise increased inter-lobular connective tissue. The picture is a peculiar and rare one. The liver-cells are so isolated that the general aspect of our liver section under the microscope is not unlike that of cartilaginous tissue, only a less regular distribution of the cells. Further, it is seen that this sparseness of liver-cells is due to the latter being partly atrophied and partly directly pushed asunder by the mentioned development of inflammatory connective tissue. Only in some places an indication of the lobules can be made out by the portal vessels and the hepatic arteries. In some fields connective tissue only is seen, in others small groups of liver-cells. The remnants of a liver lobule may be recognized. The bloodvessels are mostly empty; their calibre is diminished, or even obliterated in many places. The muscular wall of all larger vessels is attenuated, while the adventitia is rather thickened. The bile-ducts and its capillaries are catarrhally inflamed. In character the overgrown connective tissue, which is of the dense fibrillar and areolar variety, appears like one of old standing. The liver-cells proper are highly granular, so that the nuclei cannot be recognized; the polyhedral shape of the cells is lost. The cells are mostly atrophied and disfigured; some are stretched and elongated-like, from lateral pressure. Many of the cells, and even larger areas of liver, were deeply pigmented with bile. Fatty infiltration was observed nowhere.

In addition to the microscopical appearances in this very complete report, in many fields within the lobules *new* ducts could be seen, both in transverse and longitudinal section. These ducts were lined with nucleated epithelium in all instances. Their mode of origin could not be determined.

The symptoms of the so-called hypertrophic cirrhosis are generally even more decided than that of the atrophic form, and cover a much longer period of time. Jaundice being generally the exciting cause, precedes any other symptoms. These are dyspeptic at first, accompanied by pain in the region of the liver. With the pain the liver enlarges, but the outline is smooth and hard. The spleen enlarges almost invariably. Ascites is not the rule, but occurs. Hemorrhage from the nose is spoken of as a frequent symptom. The duration of the disease is seldom under two years, and may last as long as twelve. Death is generally preceded by coma. The clinical notes of the preceding case correspond very closely to the summary of symptoms. Could it not be said, however, that, save the increase in the size of the liver, there are many cases of atrophic cirrhosis which present the same symptoms also? If one hundred cases of cirrhosis of the liver are analyzed about one-third will be found to agree well with the usual clinical and anatomical description. The remaining two-thirds can be divided into many varieties, clinically speaking, and one of these would surely include cases corresponding to the above case.

Referring to the macroscopical appearances, one finds that the liver is increased in size, the surface smooth, the capsule opaque and thickened, while the cut surface has a finely granular appearance, and single lobules are inclosed in new connective tissue. A microscopical examination of the organ reveals changes similar to those which have been described in the case just recorded—intra-lobular connective tissue and abundant canaliculi.

There are some, especially the German physicians, who deny to pathologists the right to call this a distinct disease. Arguing from the case recorded to-night, one would think it a matter of fine distinction to separate it from, to say the least, interstitial hepatitis. It is impossible, for instance, to find much difference between it and the case (No. LIV.) recorded by Murchison in his classical work, in the chapter on the latter disease. Clinically, the case appears to be one of ordinary cirrhosis, and, to the naked eye, the liver would be classified as of that affection. Histologically, however, the lesions of the atrophic form are found, and more, too, new ducts (probably) and intra-lobular connective tissue.

It is to be remembered that the term biliary is applied to those forms of cirrhosis which are due to long-continued obstruction of the duct, while the term "hypertrophic" is applied to a similar hepatic change due to alcoholism or to previous infectious diseases (Thierfelder); and that both present the same histological appearances.

Now opposed to the occurrence of biliary cirrhosis, is the fact that in the many cases of obstructive jaundice, while there is generally slight nuclear excess, it is the exception to find the change of this special disease. It is true that Legg,[1] by tying the common duct, has found that such changes take place, but the control experiments of Litton entirely oppose his conclusions. It is rational to suppose that irritation of the ducts would start cell-proliferation. The remarks of Friedlander, however, that epithelial worry is more likely to lead to atypical development of epithelial cells, is just as rational and probably more frequently found in practice. Do the histological appearances have any special significance? The apparently new development of ducts is not peculiar to this affection, but is found, according to Goodhart, in acute yellow atrophy, in the nutmeg liver, and in cancer and tubercle of that organ, and in cirrhosis, especially in its active stage. While, on the other hand, the intra-lobular overgrowth of connective tissue is found not only in hypertrophic or biliary cirrhosis, but also in the hepatitis of congenital syphilis. Woodhead has a plate in his new book, illustrating a section of the latter disease, which could be taken for one of hypertrophic cirrhosis.

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[1] Legg, op. cit., p. 341 et seq.

The appearances which seem to indicate an increase of the biliary canals has been explained in many ways. Time will not permit an examination of these questions. An admirable *résumé* of the various views by Goodhart may be found in the Sydenham Society's publication. It may be remarked, however, that the simplest and more plausible explanation is that of Brüger. He considers the appearance to be due to the bile capillaries, which are rendered more distinct by the atrophy of the liver cells, and we would add, *by previous dilatation from obstruction.*[1]

To conclude, one is warranted in saying this and no more, that there are several affections of the liver which appear to have an arrangement of the cells simulating biliary canals, and hence that there is a new development of these canaliculi, or an increase in their size; second, that there are some affections of the liver (mono-lobular cirrhosis, and congenital syphilitic hepatitis) which show an increase of connective tissue within the lobules (intra-lobular); third, that the connective tissue is practically a lifeless one, and hence does not undergo contraction and reduction in the size of the organ; fourth, that rarely, in some forms of alcoholic hepatitis and in hepatitis secondary to biliary obstruction, these histological changes are presented. With such data, would one be justified in dignifying such a combination of changes with a new name?

While they are said to differ clinically, it seems that this difference is only relative. In the first place, jaundice must be excluded as a symptom, as it is the effect of the cause of the hepatitis, developing *pari passu*, or is a cause itself. Then again, if we take a large series of cases of cirrhosis, we would be enabled to arrange them into many classes from the varying symptoms, and doubtless many would conform, in symptoms, to hypertrophic cirrhosis. Actually, enlargement of the liver and of the spleen, and the long duration of the disease, are the only distinctive symptoms. And even the latter is relative, for who can tell how long an atrophic cirrhosis had been in progress? For accurate diagnosis, one would say, if in a case of jaundice we have sequential enlargement of the liver and spleen, there is, in all probability, that form of interstitial hepatitis which has its prototypes in congenital syphilitic diffused hepatitis.

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[1] The writer has recently studied with Dr. Hughes sections of a liver which had undergone the changes of a hepatitis, due to congenital syphilis. In these sections the appearance of new ducts was very distinct as well as the decided increase in intra-lobular tissue—presenting in fact the histological change of a hypertrophic cirrhosis.

In the *Transactions of Path. Soc. of Phila.* (vol. viii.) a case is recorded by Dr. Guiteras, which the writer had the pleasure of studying with him. It was diagnosed during life as "chronic catarrhal jaundice, resulting in hyperplasia of the connective tissue." The jaundice was of ten months' duration, the liver and spleen were enlarged, ascites was absent, emaciation and gastro-intestinal symptoms were marked—clinically representing a true biliary cirrhosis, yet on microscopical examination of the liver, it was found that the connective tissue in the inter-lobular spaces alone was decidedly increased, and infiltrated with embryonic cells.

Such frequently conflicting clinical records and histological changes only go to show that only the most liberal construction can allow one to evolve a new disease out of such heterogeneity. The writer is one of those who believe that its close alliance to the ordinary sclerosis forbids one giving hypertrophic cirrhosis the distinction of a separate place in nosology.

## ARTICLE XIII.

## A CASE OF LEFT INGUINAL COLOTOMY FOR IMPERFORATE RECTUM; RECOVERY.

By W. H. HAYNES, M.D., of New York.

ON the night of September 5, 1882, I was summoned to see the fourth child of Mrs. A., who had been born on the afternoon of the 3d, in consequence of its not having had a stool since birth. Upon arriving at the house, I found a male child apparently not suffering, and without any external marks of abnormality. Domestic cathartics had been administered without success, and the midwife, on attempting to give a warm soap-water enema, found that the nozzle of the syringe could be introduced within the anus only for a short distance, and the fluid injected immediately returned without causing a fecal evacuation. Proceeding to examine, I found I could only introduce the tip of my little finger within a naturally formed anus for about half an inch, into a cul-de-sac to which there was but one opening—that of entrance. Nothing distinguishable could be made out beyond the limiting membrane in the abdominal cavity. In other respects the child was perfectly natural; nursed, did not vomit, slept, and urinated. The condition of affairs was explained to the parents, who were warned against using any evacuant measures, but, if necessary, to administer small doses of paregoric p. r. n.

The next morning the child was seen by Prof. Van Buren, who gave the opinion that the best procedure in this case was left inguinal colotomy. That night the child was restless, cried, and vomited a little curdled milk.

On the 7th of September, just before noon, in my office, assisted by Drs. Hathaway, Church, and Busche, the child being four days old, was